223 ELECTROPHYSIOLOGICAL (EP) EFFECTS OF D-SOTALOL ON HYPOXIC MYOCARDIUM. Steven M. Yabek, Rinya Kato, Bramah N. Singh, Wadsworth VA Hospital, Los Angeles & Dept. of Pediatrics, University of New Mexico, Albuquerque.

The EP effects of acute hypoxia (H) are probably responsible for many clinical arrhythmias (A) in children. Sotalol is a potent anti-A agent with classII (β-blocking) and ClassIII (Amiodarone-like) actions. Its D isomer (DS) should be devoid of β-blockade & be a pure ClassIII agent. We evaluated the cellular EP effects of DS on acutely hypoxic myocardium using rabbit atrial muscle and standard microelectrode techniques. Following control action potential (AP) recordings, tissues were exposed to 10^{-4}M DS, acute H, or DS+H for 20 min. Continuous AP recordings showed no changes in maximum diastolic potential or Vmax from any intervention. The effects on AP amplitude (APA), AP duration (APD) at 50 and 90% repolarization and atrial effective refractory period (AERP) are shown (*p<0.05; +p<0.01):

	APA (mv)	APD50 (ms)	APD90 (ms)	AERP (ms)	AERP/APD90
Control (n=17)	97±4	36±9	72±9	77±14	1.08
	% CHANGE FROM CONTROL				
DS (n=5)	+2±9	+12±11*	+21±7+	+25±16*	+2.8
H (n=6)	-6±5*	-34±14+	-21±13*	- 9±7+	+26.0*
DS+H(n=6)	-7±11	-35±13+	-22±13+	- 5±19	+22.0*

By prolonging AERP and APD proportionally, DS showed typical ClassIII activity. Its acute onset and lack of antiadrenergic effects provide significant advantages over Amiodarone. DS does not, however, alter the EP effects of acute H and may be of little benefit in treating A induced or exacerbated by H.

224 ECHOCARDIOGRAPHIC LOCATION OF THE INTERATRIAL SEPTAL COMMUNICATION IN INFANTS WITH HYPOPLASTIC LEFT HEART SYNDROME, Scott B. Yeager, Alvin J. Chin, Stephen P. Sanders. (Sponsored by Barbara Jones). The Children's Hospital, Boston, Mass.

Subxiphoid two dimensional echocardiograms (echos) were examined in infants (1 year) with hypoplastic left heart syndrome (HLHS, n=15), secundum atrial septal defects (ASD2, n=15) and persistent fetal circulation (PFC, n=15). The location of the interatrial communication (IAC) was characterized in horizontal (H) and sagital (S) echo planes. Measurements determined were: 1) H plane distance from the center of the IAC to the right atrial posterior wall (H_C), 2) total H plane atrial septal length (H_L), 3) S plane distance from the center of the IAC to the superior right atrial wall (S_C), and 4) total S plane atrial septal length (S_L). Results were expressed as ratios (mean ± SD):

	HLHS	ASD2	PFC
H_C/H_L	.30 ± .10	.48 ± .10	.54 ± .08
S_C/S_L	.24 ± .09	.54 ± .09	.59 ± .09

	H_C/H_L	S_C/S_L
HLHS vs ASD2	p<.001	p<.001
HLHS vs PFC	p<.001	p<.001
ASD2 vs PFC	ns	ns

Thus, the IAC in HLHS is more posterior and superior than in ASD2 or PFC. This observation may have implications for catheter manipulation and atrial septosotomy, as well as providing insight into embryogenesis of HLHS.

225 DIAGNOSIS OF PULMONARY HYPERTENSION IN CHILDREN WITH ENDOCARDIAL CUSHION DEFECTS BY M-MODE ECHOCARDIOGRAPHY. Ming-Lon Young, Pedro L. Ferrer, Arthur S. Pickoff, Dolores Tamer, Grace S. Wolff, Otto Garcia. University of Miami School of Medicine, Department of Pediatrics, Miami, FL.

Selective Echo parameters have been used in separating children with VSD with and without pulmonary artery hypertension (PAH). This study was performed to assess specific Echo parameters in pts with endocardial cushion defects (ECD) with and without PAH. 22 pts (2 months - 11 years, 17 females/5 males) were compared with 27 normal controls (C). 5 pts had ASD I°, 3 pts had VSD of the ECD variety and 14 pts had complete A-V canal. Pts were divided into Group I (GI) 4 pts with pulmonary artery systolic pressure (PASP) <50 mm Hg; (GII) 18 pts with PASP ≥50 mm Hg. Differential tricuspid-mitral closure (ΔTc-Mc) was: (C) = 22 ± 8 msec, (GI) = 34 ± 12 msec, (GII) = -2 ± 15 msec (p < 0.001 as compared either with (C) or (GI)). ΔTc-Mc ≤10 msec was found in 0/4 pts of (GI), 15/18 pts of (GII). Right isovolumic contraction index (RICI = right ventricular pre-ejection period minus Q to tricuspid closure interval) was: (C) = -4 ± 9 msec, (GI) = -1 ± 9 msec, (GII) = 22 ± 18 msec (p <0.001 as compared with either (GI) or (C)). RICI ≥10 msec was found in 0/4 pts of (GI) and 13/18 pts of (GII). Utilizing both values of ΔTc-Mc ≤ 10 msec and RICI ≥10 msec, 17/18 pts with PAH of >50 mm Hg were recognized with no false positive. Thus, these two parameters (early tricuspid closure and increased right isovolumic contraction index) are useful in detecting PAH in children with ECD and could be used to optimize the time of cardiac catheterization.

226 DEVELOPMENTAL CHANGES IN THE END-SYSTOLIC PRESSURE DIAMETER RELATIONSHIP (ESPDR) IN PUPPIES.

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In order to evaluate the developmental changes in the ESPDR, an index of ventricular contractility independent of afterload and preload, we studied 13 normal puppies age 6 weeks to 6 months, weighing 1.7 to 21.0 kg (mean 6.8 kg). Arterial blood pressure and M-mode echocardiographic left ventricular dimensions were measured simultaneously during brief balloon occlusion of the inferior vena cava. The ESPDR using this technique was linear (r=0.95±0.02) over the range of end systolic pressure from 104.9±22.0 to 75.7±23.5 mmHg. The slope of the ESPDR (E_{es}) correlated significantly with the left ventricular diastolic diameter (LVD) prior to balloon occlusion, (r=-0.63 p 0.02) with E_{es} = -31.3 LVD + 154. The diameter intercept, D_o, did not correlate with LVD (r =0.33). We conclude that E_{es} does decrease with normal growth and the resultant increase in LVD. This apparent change in E_{es} with growth may be normalized by the LVD, suggesting that left ventricular pump function and contractility does not change in puppies over the ages studied. Furthermore, studies of the ESPDR in pathological states which alter the LVD should normalize E_{es} for LVD to more accurately assess left ventricular contractility.

227 EFFECT OF PROPRANOLOL ON THE END-SYSTOLIC PRESSURE DIAMETER RELATIONSHIP (ESPDR) IN PUPPIES.

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The ESPDR determined by pharmacologic changes in afterload has been used to assess left ventricular (LV) function in the intact animal. In order to avoid alteration of autonomic cardiac tone and thus, myocardial contractility induced by pharmacologic alterations of LV afterload, we used balloon occlusion of the inferior vena cava (IVC) to decrease LV preload and measure the ESPDR. Eight puppies 6-8 weeks old weighing 2.9±0.8 kg, were instrumented with a catheter in the descending aorta and a 1.5 cm balloon occlusion catheter in the IVC. LV end systolic diameter was measured with M-mode echo. Five-second occlusion of the IVC resulted in a fall of the end-systolic pressure from 93.9±8.0 mmHg to 63.7±10.7 mmHg, with no significant change in the heart rate (191±32, 193±35 BPM). The ESPDR was linear (r=0.96±0.01), with a slope (E_{es}) of 98.5±34.1 mmHg/cm and a diameter intercept -0.02±0.31 cm. Administration of propranolol (0.1 mg/kg) significantly decreased E_{es} (64.8±12.6, p < 0.05). In puppies, preload alteration by E_{es} balloon occlusion yields ESPDR eliminating reflex changes in cardiac tone induced by pharmacologic manipulations of afterload. It demonstrates propranolol induced changes in contractility, and thus, beta-blockade should not be used routinely in assessing the ESPDR.

228 DOPAMINE(DA) INFUSION: EFFECTS ON HEMODYNAMICS AND CATECHOLAMINE (CA) CONCENTRATIONS. Arno L. Zaritsky, Murray M. Pollack, Deborah H. Schaible, C. Raymond Lake (Spon by G. Rosenquist) GW Univ Sch of Med, Children's Hosp Nat Med Cntr, Departments of Peds, Anesth; Washington, D.C.

This study evaluates the effects of graded DA infusions on hemodynamics and plasma epinephrine (E), norepinephrine (NE) and dopamine (DA). Five children with stable blood pressure but requiring inotropic support were studied. Two sets of baseline (BL) plasma CA and hemodynamic variables were obtained; patients then received dopamine infusions at 5, 10, 15 and 20 μg/kg/min for 20 minutes each. Data sets were obtained at the end of each infusion. Measured hemodynamic changes were compared to BL by analysis of variance and changes in CA concentrations were analyzed by linear regression. Results: Significant (p < 0.05) hemodynamic changes include: a decrease in mean arterial pressure at 5 μg/kg/min; increased right ventricular stroke work and O_2 availability at 10 μg/kg/min; increase in cardiac index and left ventricular stroke work at 20 μg/kg/min. There were significant (p <0.01) linear correlations between DA infusion rate and plasma E (r = 0.68); NE (r = 0.84) and DA (r = 0.94). E and NE increased from mean BL concentration of 251 and 371 pg/ml to 753 and 679 pg/ml respectively at 20 μg/kg/min of DA. Conclusions: 1) DA infusions produce variable hemodynamic effects in patients emphasizing the need to individually titrate the infusion rate. 2) DA induced significant increases in plasma E as well as NE. This suggests that the hemodynamic action of DA may reflect increases in plasma E along with the previously recognized DA-induced NE release.